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EDITORIAL.

Penalties of Progress.

IT is a sad reflection, but it appears to be a true one, that every step forward in material affairs has a corresponding drawback. We improve transport beyond all imagination, and we pay for it by an immense increase of deaths on the road. We telephone and telegraph, and the gentle art of letter-writing is abolished. Dozens of similar examples might be brought forward. Among them would be found the art of anæsthetics. The improvements introduced by mechanisation of the administration are no exception to the rule that material progress is accompanied by counteracting disadvantage. The long operations rendered possible only by modern methods of getting and maintaining anæsthesia are accompanied by post-operative fatalities from which simpler and shorter procedures were free. The latest addition to those disadvantages which accompany progress in anæsthetics is explosion. With simple methods explosion was unknown, unless gross carelessness combined with open flame or fire. Machinery for administration has

brought about conditions in which explosion occurs with much greater ease. Hitherto it appeared that the humid atmosphere of this country kept us free from most of this particular danger, as it has shown itself in America. Recently we have had terrifying evidence that the British anæsthetist can no longer believe himself immune. And once more it is progress which has introduced the danger. It is the improved air, the dried and warmed atmosphere of the up-to-date theatre which has rendered possible the static spark which explodes machines. There is no need to point out the obvious moral. Progress is not complete unless it includes its own safeguards.

THE IDEAL GENERAL ANÆSTHETIC

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ALMOST from the time when the anæsthetic properties of "sulphuric ether" were first put to such humanitarian and practical use by Dr. Long in Kentucky, physicians and surgeons have looked forward to the discovery or creation of *the ideal general anæsthetic.* The properties essential to this ideal anæsthetic are generally considered to be: (*a*) safety, (*b*) ease and comfort of both induction and recovery, (*c*) ability to relax the voluntary muscles in narcosis, and (*d*) simplicity of administration. The general opinion seems to be that in the future some super-chemist will create or discover some substance which will with entire satisfaction meet all of the postulated demands of our ideal anæsthetic. In thus awaiting and searching for this ideal anæsthetic we are like the man in the old fable who travelled many weary miles in search of happiness only to return home to find happiness on his own doorstep. We are using the ideal anæsthetic every day without realizing its potentialities for ideal or nearly ideal anæsthesia. I refer to ether.

When administered by modern methods of vapour-oxygen technique, ether can be made to give entire satisfaction to both surgeon and anæsthetist. The patient who undergoes narcosis still, in spite of recent advances in the mode of administration, can find much to his dissatisfaction. In this regard if we, as anæsthetists and surgeons, can eliminate nausea and vomiting from ether anæsthesia, we have right at hand our wished-for ideal general anæsthetic. At the present time its apparent failings are really our failings as anæsthetists and surgeons. Ether as a general anæsthetic is perfect if we thoroughly appreciate its proper administration *to a scientifically prepared patient.*

When one considers the care with which athletes are usually "trained" for comparatively unimportant athletic

competitions, the nonchalance and casualness that usually accompanies the preparation of our patients for major surgical operations is entirely "out of line." In the great majority of cases requiring elective major surgery the patient receives no preliminary preparation whatever. She enters the hospital the evening before the day of operation, and all too often the novelty of her surroundings and apprehension regarding the approaching operation banishes that sleep that is so essential to her future well-being. On the day following admission to the hospital she is subjected to a severe test of mental, physical and biochemical endurance in preparation for which she has had no preliminary "training." As surgeons we take too much for granted when we assume, as we do, that any individual who is in apparent average health is a good operative risk for immediate elective surgery. It is my studied opinion that many patients who die following elective surgery, or who recover only after a stormy post-operative course, are thus endangered by lack of what may well be called "biochemical balance." This view is rather radical and requires further elaboration and discussion. It will be recalled that my theme is that ether, preceded by adequate and scientific pre-operative care, proves to be an ideal general anæsthetic.

Observations by experienced physiologists and practical clinical experience and experimentation will be drawn upon to lend support to the above opinions.

Prof. Dragstedt has proved that the tetany and death that usually follows in untreated thyro-parathyroidectomized dogs can be entirely prevented by simple pre-operative dietetic measures. If the dog be put upon a meat-free diet, rich in lactose and calcium, for about one week prior to operation (until such time as his solid excreta are a light brown in colour) he can be thyro-parathyroidectomized (the thyroid is always removed in doing a parathyroidectomy) without developing tetany. A high calcium diet alone is ineffective in preventing post-operative tetany in these thyro-parathyroidectomized dogs.

In the light of Prof. Dragstedt's experiments one can conclude that pre-operative diet alone will protect dogs from the severe biochemical disturbances that usually follow

thyro-parathyroidectomy. It is of interest to note that the dietetically treated and post-operative symptomless dogs can be easily thrown into convulsions by a high-meat diet. It is also of more than passing interest to recall that this property of meat, whereby its ingestion brings on terminal symptoms and exitus is also manifested in adrenalectomized or hypophysectomized dogs. The rationale of Prof. Dragstedt's pre-operative dog diet is as follows: The low-meat and high-lactose high-carbohydrate diet causes a radical change in the dog's intestinal flora; the predominating *proteolytic* type of toxin-forming bacteria are superseded by the comparatively innocuous lactic acid producing *aciduric* type of bacteria. Theoretically, with this change in bacterial flora in the intestinal tract there occurs less toxin production and therefore the liver's burden of detoxication is correspondingly decreased. As a result of this decreased hepatic burden and assisted by adequate supplies of available calcium, the dog is able, unaided, to successfully withstand such a radical procedure as thyro-parathyroidectomy.

The role of calcium as an assistant to liver detoxication is well illustrated by the researches of Dr. P. D. Lamson. The latter experimented in the production of lethal effects in dogs, using carbon tetrachloride as the poison. At the start of his investigations he was on the staff of Johns Hopkins University in Baltimore. Here he found that an average dose of 252 c.c. of carbon tetrachloride was fatal to a dog. In the midst of these experiments Dr. Lamson was transferred to a university in Nashville, Tennessee. On continuing his researches in carbon tetrachloride he found, to his surprise, that Nashville dogs of weight and breed similar to the Baltimore dogs succumbed to a much smaller dose of poison. Subsequent inquiry showed that, whereas the Nashville dogs were fed on meat only, the dogs used in the Baltimore experiments were fed on meat *and bones*. Dr. Lamson showed, by adding calcium to the food of the Nashville dogs, that the Baltimore dogs owed their increased resistance to the toxic effects of carbon tetrachloride to the high calcium content of their diet. This observer also showed that dogs could be brought out of carbon tetrachloride convulsions by the administration of intravenous

calcium chloride. These experimental observations indicate that calcium assists the body in its vital function of detoxication.

In the days preceding the routine pre-operative ingestion of Lugol's solution by patients requiring thyroidectomy for toxic goitre, post-operative mortality was high. The timely pre-operative exhibition of iodine has greatly increased the safety of thyroidectomy. It is highly probable that many surgical patients have an unsuspected and symptomless thydroid dysfunction. In any case it would seem reasonable that the pre-operative ingestion of iodine would possibly be beneficial to all surgical patients. This has been tried in the gynæcological service of the Vancouver General Hospital as a routine in a series of twenty cases under ether anæsthesia. The post-operative nausea and vomiting was markedly less in this series than in a control series of untreated cases.

It is now a well-established experimental and clinical fact that nausea and vomiting follows a certain degree of insufficiency of the adrenal cortical function. Another series of patients were given suprarenal cortex (Armour) by mouth prior to operation. In the patients that received an adequate dose of desiccated adrenal cortex (gr. 12 to 15 daily for four pre-operative days) the results were remarkable. Not only were they practically free from nausea and vomiting following 90 to 120 minutes of ether anæsthesia, but in several cases exhibited a remarkable degree of well-being post-operatively.

As a side observation in the above described clinical series, it was noticed that by the simple procedure of three or four days' hospitalization before operation the patient's operative and post-operative course was much smoother than the course of an individual undergoing a similar operation by the same surgeon where the usual custom of entering the hospital the evening before operation was followed. There can be no reasonable doubt that the physical and environmental equilibrium that is obtained by three or four days' rest in hospital before operation pays handsome dividends in the form of a smoother operative course and a more rapid and satisfactory post-operative recovery.

Ten years ago "ether pneumonia" was a synonym for post-operative pneumonia. With the increased use of spinal anæsthesia it has been found that post-operative pneumonia is as common in the latter type of block anæsthesia as it is under another narcosis. It would seem that the incidence of post-operative pulmonary infections is influenced by such factors as the season of the year, the nature of the operation, and the general condition of the patient. It is of the latter in relation to post-operative pulmonary infections that I wish to make a few remarks. It is well known to clinicians that resistance to infection varies in different individuals and is not necessarily related to physical strength. Recent work in vitamin research indicates that, in animals at least, lack of vitamins A, B and D definitely lowers the animals' resistance to infection. In fact, vitamin A is often called the anti-infective vitamin. It is quite probable that many patients who submit themselves for elective surgery, like many members of the body politic in winter time, are unknowingly suffering from a degree of avitaminosis, and therefore have a lowered resistance to infection. Another point that is seldom considered in connection with post-operative respiratory infection is the incubation period of the infecting organism. Mr. A. walks into the hospital on Sunday evening and is slated for operation at eight o'clock on Monday morning. There is every reason why he might develop a respiratory infection on Tuesday which he contracted on Sunday prior to admission to hospital.

Heretofore no mention has been made of the technique of ether administration as a factor in the prevention of undesirable post-operative sequelæ such as nausea and vomiting. It is a shameful reflection upon the medical profession in general that too often the administration of the ether anæsthesia is routinely entrusted to persons totally indifferent to and incompetent in its proper usage. The practice of pouring ether on to a face mask in the presence of anoxæmia and excessive endopharyngeal and endotracheal mucus is all too common. Any administration of ether that does not include suitable and adequate premedication, a smooth and careful induction, a well-conducted and adequately adjusted operative course with ether vapour and

oxygen accompanied by interstitial or intravenous saline therapy to combat dehydration, and followed by suction removal of the ever-present endotracheal mucus, should not be tolerated in any hospital or clinic. Anæsthesia should really commence the night before operation with the administration of a suitable hypnotic or narcotic that will command sleep and ensure a well-rested patient. Pre-operative medication should be suitable and adequate so that even a "nervous" individual will face the operation with equanimity. From the viewpoint of the patient, the first stage of anæsthesia is most pleasantly accomplished by the use of rectal avertin or intravenous or oral nembutal. For practical everyday use in a busy hospital, ethyl chloride in experienced hands effects a smooth and safe induction. In the operating room the plane of third-stage anæsthesia should be adjusted to meet the changing requirements of the surgeon. All major surgical procedures are attended by dehydrating and shock-producing factors which should be anticipated by the subcutaneous or intravenous administration of physiological saline. Anoxæmia always accompanies the semi-closed administration of ether unless oxygen is added to the inspired vapour. The patient's airway must be kept open and free. No intrapharyngeal or intratracheal mucus should be tolerated. Undoubtedly the proper management of the ether administration and the removal of intratracheal mucus just before the patient returns to the ward is a very forward step in minimizing post-operative nausea and vomiting.

Theoretical considerations indicate and practical experience proves that anæsthesia with ether can be made to approximate very closely the ideal anæsthetic of our dreams if it be properly administered to patients who have been scientifically prepared for operation and anæsthesia. "Scientific preparation" includes the observation of the following important principles: (1) the alteration of the patient's intestinal bacterial flora from the usual toxin forming proteolytic bacteria to the innocuous aciduric type of bacteria by the prohibition of meat and the inclusion of lactose in the diet for eight or ten days prior to operation; (2) the inclusion of adequate quantities of the essential minerals, calcium and

iodine, in the pre-operative diet; (3) the inclusion of foods known to be rich in the anti-infective vitamins A, B and D; (4) the pre-operative establishment of mental, physical and environmental equilibrium by hospitalizing the patient for at least two days before operation.

The fact that the majority of patients survive our present methods of pre-operative preparation and ether administration is no argument for the continuance of these methods. To-day we look back in amazement to the times when patients were given a "good" cathartic the night before operation, and as a result were often dehydrated and exhausted on the morning of operation. It is quite probable, in my opinion at least, that in some future time we, or our successors, will look in retrospect with equal amazement at the present custom of allowing major surgical cases to virtually walk into the operating-room off the street.

Where the patient's post-operative well-being is at stake it should be the duty of every surgeon and anæsthetist to apply every measure that offers any degree of insurance of the satisfactory outcome of the proposed surgical procedure. Undoubtedly the next great advance in surgery will be in the realm of pre-operative care. When this occurs and when anæsthetists are trained to administer ether similarly (or in better fashion) to the methods outlined above, then will ether come into its own as the ideal general anæsthetic.

ABSORPTION OF LOCAL ANÆSTHETICS BY THE HUMAN TISSUE AND THEIR TOXIC EFFECTS

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IN spite of all the remarkable progress which has been made with different anæsthetics, and the different experiments conducted by a great number of investigators, there have been very few who have attempted to understand the manner of producing local anæsthesia by these chemicals. Hardly anybody knows exactly the destiny of the injected fluid, whether it is absorbed and brought into general circulation in the same composition as injected, or whether locally it is first changed by some unknown forces or chemical process, thus reducing it to an innocuate state, and eliminating it later.

The object of my experiments have been to investigate the destiny of different chemicals used as local anæsthetics, the manner in which they break into the human tissue, and the time necessary for their metabolism and dissemination. On the other hand, I have tried to bring to knowledge all the other observations made in this direction.

The first experiments were conducted with chemicals which could not produce any local action upon the human tissue, thus enabling the investigators to study the physical changes and influences of their solutions.

After being injected, the fluid at once commences its action. A small quantity is first transmitted to a vein or lymph vessel, where it quickly disperses into the general circulation; the remaining fluid, which is still at the place of injection, is absorbed deep into the surrounding tissue. Although no local changes are produced, differences may occur in the osmotic tension, but, in the event of this, an equalization takes place. The interesting research made by Hamburger concerning the absorption of fluids from the

serous cavities, conduces in this direction. In these regions Hamburger observed the following: If serous fluids or salt solutions are introduced into the abdominal cavity of animals they will be absorbed. If these fluids are isotonic to the blood of the test animal there is no change in the osmotic tension. During absorption in the abdominal cavity, hypotonic and hypertonic solutions become isotonic. When in the abdominal cavity the intra-peritoneal fluid mixes partially with the blood-plasm. For instance, it was found after an injection of a 1.7 per solution of sodium sulphate, which is isotonic to the blood of rabbits, that a considerable amount of NaCl, sodium phosphate and protein, was contained in the isotonic fluid. There is no retardation in the absorption of the dissolved substance if the osmotic tension is different from that of the blood-plasm. Hamburger emphasizes the fact that the equalization of the difference of the tension independently takes place during absorption. This is of practical interest owing to the wrong belief that cocaine is absorbed slowly in hypertonic solutions, and that for this reason it is not advisable to utilize them.

Interstitial absorption of a subcutaneously injected solution reveals little difference from absorption in the abdominal cavity. Independent from the stream of water which causes a shrinkage or swelling of the tissue, a mixing of the molecules and the substances present in the solution and tissue fluid occurs. Even osmotically active saline will in this respect be entirely indifferent, because the tissue fluid contains other salts in addition to NaCl. Hamburger discovered that red blood-corpuscles supply a saline solution with haemoglobin; the amount which disseminates and surrounds the place of injection depends upon the concentration of the solution and the speed in diffusion of the substance, which itself is influenced by the permeability of the membranes and tissues. If the injected fluid is under a high pressure it reaches the surrounding tissues by means of a simple filtration; a process by which, finally, most of the dissolved substance as well as the fluid reaches the circulation. The absorption is certainly a living process; it is bound to the activity of living tissue, slow in a low

metabolism, quick in an increased metabolism, and abolished as long as the process of life is discontinued in the tissues. It may be regarded confidently that the solutions absorbed from the subcutaneous tissue attain the blood-stream without passing through lymph-vessels (Magendie, Lebkuchner, Asher, Munk, Hamburger), where, on the other hand, oily solutions are absorbed much slower by the lymphways.

The Toxic Effects upon the Human Body.

There are only very few inactive solutions, in other words, solutions which have only a physical action upon the living tissue. In comparison with the countless number of substances which cause not only physical but mainly chemical changes in the tissue with which they come into contact, these changes could have been interpreted as local poisoning to the tissue in which they are injected, their action is very different; they either increase, inhibit or abolish the function of the organ, irritate or paralyse the sensitive nerves, being even able to damage the tissue to such an extent that it dies. Their action is transient; the human tissue through some unknown forces is able to eliminate these substances, particularly when they derange the function of the organs, thus threatening their life. The living tissues later reassume their normal functions without any permanent damage. However, this is not always the case; frequently the local action of different chemical substances cause permanent changes, resulting in more or less extensive damage of the tissue. Some of the substances cause blood congestion in the field of action, others do not change the blood-supply at all; a few of them contract the blood-vessels, thus minimizing the amount of blood in the tissues or even emptying them completely. Finally, there are substances which, when coming into contact with the tissue, producing immediately local oedema, a symptom which is similar to the prick of an insect, or even inflammation or necrosis.

Absorption of Different Anæsthetic Solutions.

The process of absorption of different anæsthetic solu-

tions differs completely from those of the inactive materials. As a rule a local action can only occur if the active material is partially bound to the tissue or organ where injected, and thus it cannot be absorbed immediately. At the present time we are still in doubt as to the further fate of the active material. It has been proved that some alkaloids, such as cocaine and adrenalin, are not absorbed in their natural form, and in order to free them the body decomposes them. A substance absorbed slowly from its place of application, diffuses deeply into the tissue having a stronger local action, than if it had been transported by immediate absorption from the place of application. We shall learn that the artificial retardation of absorption is a great help in local anæsthesia. When different substances chemically come into contact with sensitive nerves they produce, without exception, an incidental or permanent paralysis—that is, anæsthesia which is preceded by a strong irritation. A few substances have been discovered, the application of which causes local poisoning with transient sensitive paralysis without irritation or damage to the tissue; these are the substances which are suitable for local anæsthesia.

The test-methods which attempt to ascertain the ability of the local anæsthesia, suffer from the fact that the ability to produce it depends, to rather a high degree, on the type and place of application. The first examination in this direction came back to the idea that general narcotic drugs, in contact with a nerve must have similar action so that they may be brought by the blood-stream to the brain. It has been shown that ether and chloroform when acting in fluid or gaseous form on a nerve, are able to interrupt the transference. (Longet, Bernstein, Ranke.) Many other non-narcotic substances have the same qualities, and the function of the nerve depends on its being soaked by a fluid of a certain composition.

Leibreich, Bussenius, Mullerheim and Kunowski were the first investigators to test large amounts of chemicals as to their ability to produce local anæsthesia. The chemicals were dissolved and injected subcutaneously or put into the conjunctiva of frogs, rabbits or guinea-pigs. The conjunctiva was punctured or irritated in order to discover whether

any pain was experienced. (Turck's test). On account of its low sensitiveness and distance from the surface, subcutaneous tissue is unsuitable for such tests. Although the method of these authors were, to a certain extent, imperfect, they found that the majority of narcotic and non-narcotic substances do not leave the sensitiveness intact, but, according to Leibreich, they are *anæsthetica dolorosa*, which means that they produce anæsthesia after a period of irritation.

Heinze discovered, by means of the first sensation produced by the penetration of the fluid, that there are only a few chemically inactive substances which do not disturb the sensitiveness when brought into contact with a sensitive nerve. The majority of them are active, only a few have the ability to inhibit the function for a short time, without either irritating or damaging the tissue. This sensation, when experienced by the human body, presents the actual value of the local anæsthetic power of one substance when compared with another.

The first value results from the ascertainment of the lowest concentration of the solution where a local action can still be observed. This procedure is accomplished by testing a solution of various degrees. The lower the limit of action, the higher must be the affinity of the substance to the tissue. All our local anæsthetics influence the nerve parts in an extraordinary high dilution.

The second value is ascertained by injecting into the same person at the same time, equal amounts of the same concentrated substances, in this way testing the time during which sensibility is absent. The longer this is the case, the more stable must be the changes the substance produces in the nerve. Actually the time of anæsthesia depends upon many other circumstances, such as the person, the amount of blood on the place of injection, the speed of absorption, and concentration of the solution.

Other tissue changes can be observed in the field of penetration on the skin. It is always possible to observe the quick disappearance of this field which leaves no trace providing that the substance is absorbed without local damage to the tissue. In cases where the solution produces

inflammation or contraction of the blood-vessels the final result always leaves a more or less conspicuous damage, sometimes ending in necrosis. When testing codeine, morphia, peronine and ropacocaine, I found that these substances produce acute local oedema similar to an insect prick. In this way a substance may be tested easily and quickly without damage to the subject, provided the experiment is done with highly diluted solutions.

When parts of the substance are not brought direct to the nerve by the syringe, but reach it by means of diffusion, the anæsthetist depends not only upon the above results but also on the permeability of the membranes and the speed of diffusion of the substance. For instance, a substance with a great local anæsthetic power is little or not at all suitable for a certain purpose, because it cannot penetrate a membrane or tissue in its attempt to reach the nerve. Cocaine, with its high local anæsthetic power, is absolutely inactive when applied to the skin owing to its lack of power to penetrate. When employed in the same way, diluted solutions of phenol with considerably less local anæsthetic power diminish the sensibility of the skin far better. It is possible to compare different substances if the solutions of the same concentration are injected in the surrounding tissue of certain skin nerves of the subject, particularly when the nerves are superficially in the subcutaneous tissue, and also when the duration and diffusion of the anæsthesia in the region of innervation of the nerve has been tested.

It has been shown that the anæsthetic power of chemical compounds is bound to the presence of certain groups of atoms, which Ehrlich called "anæsthesiphorous groups," whereas the remaining groups may be replaced by others, thus forming new local anæsthetic substances. After Einhorn discovered the chemical composition of cocaine, thus making the synthetic production of this alkaloid possible, it was utilized for numerous interesting tests in order to combine its anæsthesiphorous group of atoms with new groups. This conscientious chemical research resulted in the invention of a number of new anæsthetics (holokain, eukaine, orthoform, later stovaine, alypin and novocaine).

This must certainly be admitted as a triumph of German science.

All the local anæsthetic substances discovered up to the present have the following qualities: they are, like the general anæsthetic, poisonous for the protoplasm which paralyse not only the nerve substance but every protoplasm brought into contact with it. One of the characteristics of these drugs is their strict affinity to the nerve tissue. They paralyse the function of the latter, even though they are in an extraordinarily high dilution; but are insufficient for a noticeable influence upon the protoplasm of another type. If a sufficient amount reaches the circulation quick enough it produces general intoxication apart from the required local action. The affinity of these substances to the nervous system explains the fact that the appearances of poison are first observed on the central nervous system.

It is of extreme importance that these general actions do not merely depend upon the dose used—narcotic substances have no maximal dose—but also on the speed of absorption.

A typical character of local anæsthetic substances is their ability to involute the changes induced by them. They are able to interrupt the nerve function temporarily without leaving permanent damage, thus differing from Liebreich's *anæsthetica dolorosa*, by the fact that they paralyse without previous irritation and that they do not cause damage to the tissue. Gross concluded his investigations concerning the general qualities of local anæsthetic substances as follows: "The bases of the substances—cocaine, novocaine, stovaine, eukaine and alypin—were examined; they are approximately the same, except that they have a far stronger action than their salts; this is proved by the quicker and better action produced in their high dilution. The different action produced by the chlorides of the usual substances depends only upon the different degree of hydrolysis of the solution. The weaker the acid formed the more complete is the hydrolysis. For instance, a novocaine bicarbonate solution has an action five times stronger than an æquimolecular novocaine chloride fluid. It was revealed that sensitive nerves are more susceptible than the motor nerves.

Gross made further attempts to make clear the process upon which local anæsthetic action is based. He commenced with general anæsthesia, which had previously been investigated by Overton and Meyer. They stated that the action of an anæsthetic depends upon its action on the lipoids of the central nervous system, and also upon the fat solubility of the drug. In other words, the anæsthetic power of a drug is proportional to its fat solubility, and in the same way could be interpreted by the relationship between the solubility of the drug in fat and water. On account of the stronger fat solubility the anæsthetic collects in the central nervous systems, where it does not enter a chemical reaction (Meyer and Overton), but only produces changes of physical nature on the lipoids. Gross states that this theory is also valuable for local anæsthetics and their action upon the peri-pheri nervous system.

Without rejecting the relations between narcotics and lipoid, discovered by Meyer and Overton, Verworn, Burker and others defend the older point of view that the narcotic action can only be explained by the formation of chemical compounds in the central nervous system. They supposed that the anæsthetics combined with the oxygen of the nervous substance, and thus produced a temporary asphyxia along with paralysis of the physiological function. The fact that in local poisoning, parts of the anæsthetic employed are destroyed and that these parts never reach the circulation, favours the chemical theory, at least as far as the anæsthetic is concerned.

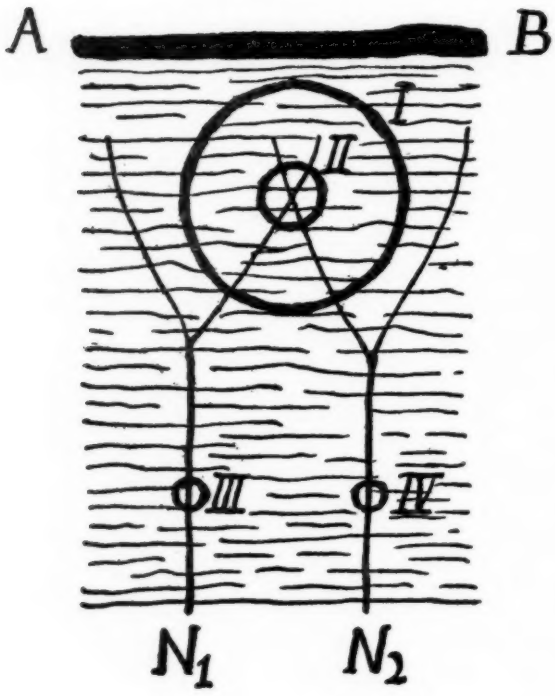
Before we consider several local anæsthetics it is necessary to know the exact way in which an anæsthetic solution acts, thus producing terminal and branch anæsthesia in man.

In order to avoid complicated explanations I should like to present here a simplified scheme given originally by H. Braun.

Mechanism of Producing Local Anæsthesia.

This schematic diagram could be used for any part of the human body, providing that it is covered by a surface AB, representing the skin, mucous membrane, serous or

synovial membrane of a natural cavity. N1 and N2 represent two sensitive terminal nerve branches which, by ending in different directions, innervate the part of the tissue in question. The terminal nerves divide in such a way as to provide sufficient innervation for all portions of



the tissue. If we intend to render Circle I of a certain tissue insensitive we must unconditionally bring all nerve branches which innervate this circle into contact with the injected fluid. Only by accomplishing this do we succeed in producing terminal anæsthesia.

This anæsthesia can be produced by different methods. Introducing a hollow needle into Circle I, a sufficient quan-

tity of an anæsthetic solution is injected under certain pressure in such a way as to render insensitive all districts soaked with this solution. By infiltrating the tissue the anæsthetic solution replaces the normal tissue fluid. The parts of the dissolved substance, pressed mechanically, come into contact with the elements of the tissue immediately after the injection, and in this way their local action is equally distributed throughout the entire district. Naturally a difference will be observed in the action corresponding to the anæsthetic used, particularly when the solution is diluted to the lowest limit of activity of the dissolved substance. This method of terminal anæsthesia is very well shown through the original field of penetration, and it is called Schleich infiltration anæsthesia. Duration and strength of the sensibility depends upon the stability of the changes occurring in the nerve tissue, and the concentration of the solution. The more concentrated the solution, the longer will the tissue infiltrated by it remain insensitive. In the entire district the abolishment of sensibility has the same duration and strength on account of all its nerve parts being caught in the same way.

Another method of producing terminal anæsthesia in District I is to inject a solution of different substances into the mid-point. For instance, in the small District II, which occupies the centre of the original circle, solutions of different anæsthetics are injected under a certain pressure. Immediately in this district local changes occur, leading to infiltration anæsthesia. Whether the solutions have an osmotic pressure different from that of the blood or not, an exchange of the parts of the tissue fluid and the dissolved substance takes place. They diffuse into the neighbourhood according to the speed of diffusion, permeability of the membranes and concentration of the solution and produce, after a certain time, the same changes in District I as at the mid-point, which stands under direct action of the drug. However, there is one difference; the solution during diffusion becomes more and more diluted the further it travels from the mid-point, by mixing with the tissue fluids. The parts of the dissolved substance which have travelled little are more active because they still have to

spread over a large area. In the periphery of the infiltration field there are only a few active parts of the dissolved substance because by spreading over a large area the majority of them have been bound to the tissue elements and consequently lost their activity. The strength of the local poisoning will decrease from the centre to the periphery of the field and a certain time after the injection of the solution District I will present different degrees of anæsthesia, the highest will be at the mid-point where the solution is concentrated and gradually decreases towards the periphery. For obtaining a uniform anæsthesia of all District I it is necessary to infiltrate a small quantity of concentrated solutions in different parts of the district. This method is called the "indirect infiltration anæsthesia."

Finally, a third method of producing infiltration anæsthesia is obtained by means of diffusion. By bringing parts of the dissolved substance into contact with the surface AB, District I could be rendered insensitive. Anæsthetic action can only occur here, if the membrane covering the surface is permeable for the dissolved substance and if the concentration of the solution is stronger than the one which should impair the sentiveness of the nerve by direct contact. This method is termed diffusion anæsthesia and it is not very popular owing to the slow development of its action.

There are many chemical compounds which are able to impair the transmission property of the nerves far from their termination. If the solutions are injected in the surrounding area III and IV of the two nerves, they diffuse into them and impair the transmission property. In this way District I is again rendered insensitive and the method is called branch anæsthesia. Solutions of a very low degree of concentration can produce branch anæsthesia, only if they are brought directly into contact with the nerve fibres. When injected again into the neighbourhood of the nerve to obtain the same action, they must be in a higher concentration and the time required for the development of the anæsthesia is much longer.

In conclusion I should like to mention that insensibility of a district can be obtained by injecting an anæsthetic

solution directly into an artery or vein, providing that the blood circulation proximally to the district which is going to be anæsthetized has been interrupted. In this way the solution is bound to spread towards the periphery and by coming into contact with the terminal nerve-branches of the tissue, renders them insensitive.

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THE ARRANGEMENT AND EQUIPMENT OF ANÆSTHETISING ROOMS

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THE introduction of new anæsthetic agents and of new methods of administration has increased the number of instruments, drugs and tools used by the anæsthetist. His equipment is now extensive, expensive and highly technical. The good old days of a handkerchief and a bottle of chloroform are now incredibly remote, and the modern anæsthetist has to have an aptitude for engineering and for minor surgery before he can start on his speciality.

Nearly all operating theatres have been arranged with a room alongside known as the "anæsthetic room." In many cases this room is ill-placed and ill-adapted for its purpose. Frequently it has only one door through which the conscious patient is wheeled in, only to be wheeled out later, unconscious, back into the corridor, and so to the theatre. He may have to pass both theatres and sterilizing room on his conscious way, and is almost certainly exposed to the risk of meeting some earlier patient being returned to the ward. It frequently happens that every sound in the theatre and sterilizing room is clearly audible and even magnified in the anæsthetic room. For these inadequacies we must either blame the architect or bemoan the lack of space or money at his disposal.

Much more serious, because much less necessary, is the way in which these rooms are looked upon as anything but the private domain of the anæsthetist. It is true that here anæsthesia is induced, and that here the anæsthetic stock is kept—the bottles and masks in a cupboard, and the cylinders in an irregular heap on the floor. But here is stored all apparatus not immediately required in the theatre, from the Horley table to the spare jar of vaseline. Here

the anæsthetist and odd visitors hang their coats and put on their gowns. Through here the theatre nurses may slip out to the telephone in the corridor. Here the surgeon comes for his afternoon cup of tea, dispensed in the theatre sister's tea-things, also kept here. Here the theatre porter keeps his squeegee and sweeping brush, and here, as likely as not, the hospital cat hides her kittens. In short, the usual anæsthetic room is grossly mismanaged and abused, and it is often in a fearsome, crowded and noisy den that the patient has to await his anæsthetist and the anæsthetist has to perform the most difficult part of his task.

It is our purpose to consider the ideal arrangements and furnishings for an anæsthetic room. First comes its position. It should be immediately adjacent to the theatre so that the trolley on which the patient is anæsthetized may be moved alongside the operating table with the least delay, or so that the table itself, if moved to the anæsthetic room, may be wheeled into place immediately.

The room should be at the side of the theatre opposite to the sterilization plant, and, if possible, away from the surgeons' and sisters' rooms. This will ensure that the approach from the main hospital corridor to the anæsthetic room is used almost solely for incoming patients. There should be a strictly one-way system of traffic so that by no means may the conscious patient meet either an outgoing patient, or a trolley laden with drums and dispensary stock. The noisy routine business of the theatre sroould be carried on as far away from the anæsthetic room as possible.

The size is next to be considered. When all fixtures, such as wall-cupboards, shelves, instrument-table, and wash-basin have been allowed for, there must at the very minimum be room enough for the anæsthetist to push his working table from one to the other without causing the ward-nurse to leave the patient's side. In our ideal room there will be just enough room to enable the patient's trolley to be turned round completely. More space than this is unnecessary, and a very large room may be alarming to the patient. It is true that a large proportion of patients come to the anæsthetic room either unconscious or drowsy, but there is still a number of cases in which premedication to the

somniferous degree is impossible or undesirable; and, further, the patient who is to have evipan-sodium has first to make a conscious journey to the scene of his injection.

The doors leading from the corridor to the room and from the room to the theatre should be large enough easily to admit the stretcher and should not be opposite to one another; for it often happens that the theatre nurse looks out to see if the next case is up just as that case arrives and so gives him a rapid view of the preceding operation. An alternative route from the theatre to the anæsthetic room corridor is a perfection, allowing the anæsthetist to approach his patient without exposing the theatre at all, either visually or audibly.

Sliding doors take the least space and open more completely than swing doors: they are, however, almost invariably noisy. Probably a large-size door of the ordinary hinge type with a sound latch, but no lock, is best. Automatic arrangements for closing are a thorough nuisance whilst the trolleys are being moved, and usually end up by keeping their doors persistently slightly ajar. The doors should be heavy and close-fitting to minimize the passage of noise from the theatre.

Heating and lighting should be part of the theatre system, as should be that of the approach corridor. It often happens that the anæsthetic room acts in winter as a wind-shoot through which pass cold blasts of air from the outside to the heated theatre. This is bad for patients, nurses and anæsthetists. The border-line between the theatre block and the greater world of the hospital in general should be drawn so as freely to include the anæsthetic room. Ventilation is best arranged for by an air-conditioning plant. The ideal anæsthetic room being situated between the theatre and the approach corridor will probably not have an outside wall and a top-light, the best form of illumination, will be inevitable. This skylight should not be used for ventilation. The mechanism will certainly go wrong in the end and a dangerous source of draught and rain-leakage will arise. In changeable weather a sudden cold down-draught can come in a moment, and it takes time to shut even a perfectly-working ventilator. For artificial lighting electric

bulbs placed at the corners of the skylight form undoubtedly the best arrangement, and they should be fitted with an alternative circuit in case of current failure.

The walls of our room and of the passage leading to it should be clean, smooth and painted or tinted in a light, non-aggressive colour. The old yellow-white tiles, so often seen full of fine crackles and even with occasional cracks, are among the most depressing products of human manufacture, and a plain, non-reflecting plaster or cement surface is much better in all respects. There should be absolutely nothing fixed to or hung from the walls above the level of the trolley. There are in use anæsthetic rooms which have walls partly covered with cupboards labelled "Poison," and for the rest festooned with gas-bags, face-masks, rubber-tubing, restraining-straps, douch-cans, arm-rests, and even with notices emphasizing the explosive dangers of ether and oxygen! To the theatre staff these things mean nothing, but to the apprehensive patient they may seem like the paraphernalia of the Inquisition.

The furnishing of the anæsthetising room must be of the plainest and simplest. First, there must be a mobile stand carrying four cylinders—two of oxygen fitted with fine-adjustment valves and dial-guides, and two of carbon-dioxide-oxygen mixture. Of these last two only the cylinder in use should be fitted with a rubber tube and appropriate valve keys should be chained to the stand. Next, there must be a wash-basin fitted with running hot and cold water supplied by a mixing-tap provided with an elbow-moved handle. Foot-controlled water-taps seem always to be faulty. Alongside the basin should be a marble or porcelain shelf on which stand bowls for spirit and biniodide. There must be an instrument table of moderate size and of fairly small height, preferably with a lower shelf. This, like the cylinder stand, must be against the wall, and it also must be in proximity to the wash-basin, so that the operating anæsthetist does not have to pass all round the room to reach his instruments.

Along one wall, or along part of it, should be a wooden fitting consisting partly of a tier of drawers and partly of a cupboard with sliding doors. The top level of this should

be no higher than that of the trolley. On it should stand a wooden trough with a rack containing small bottles of camphor, strychnine and coramine injections and ampoules of coramine, lobeline and pituitrin together with a supply of files. There should be a flat dish filled with spirit and contaning hypodermic syringes (two 2 c.c.'s and one 5 c.c.'s), a number of ordinary hypodermic needles and two fine spinal needles for use if intra-cardiac injection should be necessary. This dish should be covered by a flat glass lid with a handle for easy removal in case of emergency. There should be a smaller glass trough also covered to hold a supply of sterile swabs.

The usual syringes and needles for spinal and intravenous anæsthesia are not to be looked on as part of the anæsthetic room equipment, any more than is the anæsthetist's working trolley or the gas-oxygen machine he may use. If a spinal or intravenous anæsthetic is to be given, the apparatus and so forth must be brought in freshly from the theatre proper, which should be its home.

The cupboard should contain a spare bottle of ether and one of chloroform and a spare tube of ethyl chloride. It is not to be used as a store cupboard but merely as a source of supply in case of shortage. The drawers should hold a stethoscope, a sphygmomanometer, an electric torch with spare battery and bulb, a supply of wooden tongue depressors for throat examination, a spare Magill's laryngoscope, also with spare bulb and battery, and a small but comprehensive collection of Magill's endotracheal tubes. These again are strictly not ordinary stocks, they are for use in case of sudden need. There should be also a large canvas restraining-strap for use in case of maniacal patients—but put there in the determination never to use it.

For the rest, wherever there is space, a narrow shelf along the wall about two and a half feet from the ground is a great convenience to the anæsthetist who may have two or three drop-bottles and a mask to put down whilst he examines his patient. If there should be violent struggling during induction it is very helpful always to have somewhere at hand a safe spot to deposit an endangered bottle. A plain wooden stool should also be provided: there is no need for the

anæsthetist to stand whilst awaiting his patient, or for the nurse to stand whilst awaiting the anæsthetist. A metal stool such as used in the theatre need not be provided: there is no need for height adjustment, and wooden stools are much less noisy on concrete floors than are steel ones.

Final emphasis must be laid on the necessity of allowing only such things to be kept in the anæsthetising room as are essential for the induction of anæsthesia, the resuscitation of the collapsed, and the essential comfort of the patient, the nurse, and the anæsthetist.

We have not considered the stretcher-trolley, the operating table, the anæsthetist's working table, and if separate, his gas-oxygen machine, as part of our equipment. They have to adapt themselves to other needs besides those of our room and, in any case, are only temporary inhabitants of it. Throughout our arrangements we have tried to avoid all danger of irritating or of alarming the patient who is to occupy our anæsthetising room. All theatre staffs should be trained to look upon this room as a place of extreme privacy, where quietness is essential. A fairly wide experience of London operating theatres and their anæsthetising rooms has shown many excellencies, but the perfection we have tried to describe is yet to be found.

250 CASES OF SPINAL ANÆSTHESIA WITH PERCAINE ACCORDING TO THE METHOD OF JONES.

By Prof. Herman Franken,

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(Director: Prof. O. Pankow).

(Continued)

II. Jones considers the administration of hypnotics as dangerous, but this does not seem to apply to our cases of low anæsthesia. Jones fears that the paralysis of the intercostal nerves would add in a dangerous manner to the diminished excitability of the respiration caused by the narcotic. This theory is no doubt correct in cases of high spinal anæsthesia, when injecting 12 to 14 c.c. and more so between L_1 and L_2 or higher. However, in our 250 cases in which we injected 10 c.c. the "twilight sleep" of our patients never affected the respiration adversely. It is remarkable that the "twilight sleep" increases generally in depth during the course of spinal anæsthesia, a condition which I associate with the fall of the blood-pressure. There is no doubt that the narcophine-scopolamine "twilight sleep" represents a remarkable humane procedure, whilst without "twilight sleep" the psyche of the women is in many cases adversely affected through their being conscious during the operation. "Twilight sleep" constitutes, in addition, a very important factor of safety in low spinal anæsthesia. The safety of spinal anæsthesia is closely connected with the stabilisation of the conditions within the spinal canal, and specially with the constant height of the column of cerebro-spinal fluid. The research work of Hartwich has proved unquestionably that anxiety, excitement, cough, straining and crying are liable to drive the liquid quickly to the suboccipital region irrespective of its having a lighter or heavier specific gravity. The following table (III) shows the results of Hartwich's experiments:

TABLE III (Hartwich).

Spinal injection of 1 c.c. phenolsulphophthalein in decubitus: appearance suboccipitally:

after 20 minutes, no morphine	Patients strained as a result of fear and excitement.
after 20 minutes, no morphine	
after 15 minutes, no morphine	
after 60 minutes, with morphine ...	Quiet. Nothing.
after 2 hours, with morphine	Quiet. Nothing.
after 2¾ hours	Moribund, practically pulseless. Nothing.
after 4½ minutes	Asked to cough and strain.

Spinal injection of stained meningococcus serum (of higher specific gravity) in decubitus: appearance suboccipitally:

after 20 minutes	Child cried at intervals.
after 15 minutes	Child cried.
after 11 minutes	Child cried the whole time.
after 5 minutes	Child *cried lustily*.
after 5½ minutes	Child *strained* and *coughed*.

Consequently the quietness resulting from "twilight sleep" stabilises the column of cerebro-spinal fluid and prevents the anæsthetic solution injected into the spinal canal being driven in the direction of the medulla oblongata as a result of the behaviour of the patient.

III. We ask ourselves, *In what concentration is percaine still effective on the medulla oblongata?* It is obvious that a solution injected into the lumbar region must become more diluted by the time it reaches the medulla, no matter what the circumstances are which bring this about. The possibility of the solution reaching the medulla in a more dangerous concentration is greater with a highly concentrated injection than with the introduction of a very diluted solution, even when the quantity of the latter is larger.

Several authors have confirmed, both clinically and experimentally, that by direct application of the anæsthetic agent to the medulla it is possible to anæsthetise the head without fear and without endangering respiration (Koster and Kasman, Meleiro de Souza, Shimotsuma, Imagaron and Hang Kong Su). Some of those authors assume a different reactivity of the sensitive and the motor nerves

towards the anæsthetic. As a consequence of a series of similar experiments we have formed our own point of view on this subject. Already Harrison and Frank, as well as Cotui and Standard, disagree with the above-mentioned opinion.

In order to invesigate the question of concentration regarding the danger to, and general action on the respiratory centre exerted by different percaine concentrations, we used Standard's method and noted the costal and abdominal respiration and also the bood-pressure in 20/25 Kg. dogs narcotised with pernocton. The cervical vertebræ were exposed by incision, and the suboccipital puncture made to bring the solution into direct contact with the medulla oblongata.

Sixteen experiments were made, details of which are set out in Table IV. This table shows:

1. The injection of 3–5 c.c. of a percaine solution 1:1500 in 0.5 per cent physiological salt solution causes immediate respiratory paralysis and a fall in blood-pressure to zero in three to four minutes; even artificial respiration cannot delay death (Curve I).

2. With a dilution of 1:1 of this solution, injected in doses of 2–5 c.c., immediate respiratory paralysis and death appeared only in three of five animals, whereas in two animals only transitory respiratory depression with lowered blood-pressure was observed.

3. With the injection of 3–6 c.c. of a dilution of 1:2 of the original solution it was observed that 6 c.c. could still cause respiratory paralysis which, however, could be overcome by artificial respiration. In one case there was only a slight depression and in another no effect at all was registered. In the last case the blood-pressure fell as usual, but in the two other cases an unexpected and considerable rise took place, which lasted for 30 and 12 minutes respectively (Curve II).

4. In four cases, 2–4 c.c. of a 1:3 dilution of this Standard solution had no effect on the respiration, but in one case a slight inhibition of the thoracic respiration was noted. In three cases the blood-pressure was not affected, but in two

it rose for a period of 45 and 8 minutes respectively. (Experiment discontinued).

5. There is no connection between the quantity of liquid injected and the rise in blood-pressure, because 3 c.c. of physiological salt solution caused only a slight rise of 20 mm. Hg. which lasted five minutes, whereas from the details given in the Table it will be seen that a similar quantity of percaine solution causes an increase in blood-pressure of from 40 to 60 mm. Hg., which lasts from 12 to 45 minutes.

6. The behaviour of the blood-pressure and respiration has also no connection with the toxic amount of percaine injected. A dog weighing 25 lb. (11.4 Kg.) received intravenously 3, 5, 7, 10, 15 and 20 c.c. of a standard solution 1:1500 at intervals of 15 minutes, and another dog, weighing 20 Kg., received 10 c.c. of 1:1000 percaine solution three times intravenously at intervals of 15 minutes, and in each case blood-pressure and respiration were not affected.

7. Five c.c. of coramine injected suboccipitally within one minute after the blood-pressure had fallen to 80 mm. Hg. and the respiration showing a depression of medium strength (as a result of the previous suboccipital injection of a 1:1 dilution of a 1:1500 percaine solution), appreciably stimulated the respiration for a period of four minutes and also caused an increase in blood-pressure, which was maintained for 10 minutes before it fell to the level prior to the coramine injection (see Curve III). Therefore it appears that the suboccipital injection of respiratory stimulants suggested by Janossy and Kulenkampf, can be considered as a suitable therapy in threatening paralysis of the medulla oblongata (see Curve IV).

8. The respiratory failure is not a sequel of the primary circulatory insufficiency but appears immediately after the percaine solution has come into contact with the medulla oblongata; only, afterwards, the increasing paralysis of the circulatory centre causes blood-pressure to fall.

Jones's assertion that the danger to the respiratory centre decreases rapidly by further dilution of the 1:1500 solution

TABLE IV.

Percaine	Dilution	Quantity in c.c. injected subocipitally	Blood-pressure prior to injection	Effect on blood-pressure	Effect on respiration	
1:1500 in 0.5% NaCl = Standard solution	0:0	5	180	Fell to 0 in 4 min.	Immediate stoppage.	Death after 4 minutes. No artificial respiration.
	—	4	145	Fell to 0 in 4 min.	Stoppage after 1 minute.	Death after 4 minutes, in spite of artificial respiration.
	—	3	150	Fell to 0 in 3 min.	Immediate stoppage.	Death after 3 minutes. No artificial respiration.
Standard solution	1:1	5	—		Stoppage after 1 minute.	Death after 30 minutes, in spite of artificial respiration.
	—	4	220	Fell to 185 in 5 min.	Pronounced depression after 5 minutes.	Circulation and respiration spontaneously. Recovered in 15 minutes.
	—	3	140	Fell to 80 in 5 min.	Medium depression.	Circulation and respiration spontaneously. Recovered in 30 minutes.
	—	3	—		Stoppage after 6 minutes.	Death after 20 minutes, in spite of artificial respiration.
	—	2	160	Fell to 0 in 4 min.	Stoppage after 4 minutes.	Death after 4 minutes. No artificial respiration.

TABLE IV (*Continued*).

Percaine	Dilution	Quantity in c.c. injected suboccipitally	Blood-pressure prior to injection	Effect on blood-pressure	Effect on respiration	
Standard solution	1:2	6	110	Fell to 80 in 6 min.	Stoppage in 2 minutes.	Artificial respiration for 10 minutes. Recovery after 20 minutes.
	—	4	160	Rose to 210 for 30 min.	Slight depression for 15 minutes.	
	—	3	190	Rose to 230 for 12 min.	No effect.	
Standard solution	1:3	4	110	No effect	No effect.	
	—	3	140	Rose to 190 for 45 min.	No effect.	
	—	3	150	No effect	No effect.	
	—	2	120	No effect	Diminished thoracic breathing observed.	
	—	2	180	Rose to 240 for 8 min.	No effect.	
NaCl	—	3	160	Rose to 180 for 5 min.	No effect—injected against pressure.	

seems to be strongly supported by the results of our experiments. Already with a 1:1 dilution of Standard solution there were two out of five experiments which did not terminate fatally, while with further dilutions no deaths were registered. I have also seen in patients a surprising increase in blood-pressure, following the injection of the further dilutions, as shown in Table V.

TABLE V.

Duration of increased blood-pressure with percaine spinal anæsthesia.

Initial value	After 10–15 minutes	Duration
80/50	115/65	60 minutes
135/80	165/90	45 minutes
110/65	130/70	2½ hours
135/90	155/85	60 minutes
130/65	150/90 (180/115)	1¼ hours
135/75	170/85	30 minutes

Two possibilities can be advanced to explain this phenomenon; either the percaine reaches the main innervation points of the vascular system in such a dilution that it acts not as a paralysant but as a stimulant of the blood-pressure, or it paralyses antagonistic nerves so that the group which causes a rise in blood-pressure becomes predominant.

The phenomenon, established by animal experiments and clinical observations, that in spite of effective spinal anæsthesia, in no case was there a fall in blood-pressure, but on the contrary an increase, is interesting because it presents an ideal anti-shock action which is very much desired. The establishment of this fact at least suggests the possibility that the method of spinal anæsthesia can be improved at any rate to such an extent as to avoid every fall in blood-pressure. In the cases described adrenalin was given beforehand in four instances and ephetonine in two, as a prophylactic. However, in our experiments on animals, we noticed that in those cases in which these drugs had not been administered the fall in blood-pressure was not very pronounced.

SUMMARY.

1. Percaine spinal anæsthesia according to the method of Jones was extremely successful in 250 serious and prolonged gynæcological operations.

2. Only 3.2 per cent of the cases reported slight to medium headaches which, however, disappeared in a few days with the aid of head diathermy.

3. For gynæcological operations the injection of 10 c.c. of a 1:1500 percaine solution in 0.5 per cent physiological salt solution between L_3 and L_4 can be relied upon to produce anæsthesia lasting from one to two hours.

4. As a circulatory prophylactic against a fall in blood-pressure an intramuscular injection of ephetonine administered fixe to ten minutes before the spinal puncture is made, is very effective.

5. In high spinal anæsthesia "twilight sleep" is a danger to respiration, yet in low spinals for gynæcological operations it is a safety factor, as it stabilises the level of the cerebro-spinal fluid and, consequently, the injected solution.

6. The effective percaine solution 1:1500 is of such a low concentration that with further dilution the dangerous effect on the respiratory centre diminishes rapidly, as observed in our experiments on animals.

7. The direct application of percaine solution on the medulla oblongata first causes immediate paralysis of the respiratory centre, then follows the circulatory collapse.

8. The subocciptal injection of cormaine stimulates the respiratory centre even when it has been previously paralysed with percaine, so that this method appears to be suitable also in clinical practice.

9. In animal experiments it was observed that in isolated cases, in spite of effective anæsthesia, a fall in blood-pressure did not occur, but on the other hand there was a considerable increase, which was maintained for a fairly long period.

SECTION OF ANÆSTHETICS (ROYAL SOCIETY OF MEDICINE)

AT the meeting of the Section of Anæsthetics of the Royal Society of Medicine on March 1st, with Dr. H. P. Crampton in the chair, two papers were read, one by Dr. Reginald Ironside, on an ether explosion at the Royal Masonic Hospital, Hammersmith, and the other by Prof. G. I. Finch, on electrical ignition of gases.

Dr. Reginald Ironside, in describing the circumstances of the Royal Masonic Hospital explosion, said that the anæsthetic trolley had been in use for two and a half hours previously, with oxygen passing over ether. The trolley was being wheeled out of the operating theatre into the anæsthetic room when a violent explosion occurred. The ether container broke into small pieces and two bottles of ether on the other side of the room exploded about two seconds afterwards. The theatre attendant was knocked down, he and a sister were blown over, the patient fell off the trolley, and the whole room was a sheet of flame. They managed to remove the patient and send for the fire brigade, and the fire was soon under control. The floors of the corridors of the hospital were laid with rubber, and those of the anæsthetic room and operating theatre with ordinary granolithic flooring. The air in these two rooms was changed every ten minutes by a special apparatus supplying filtered air. The sterilizers were steamless, and the whole atmosphere extremely dry. Nurses and cleaners had complained of sparks from the trolleys carrying food and also those carrying patients. The trolleys were insulated by rubber wheels. The anæsthetic table was also on rubber wheels. The theories put forward by the Home Office expert were that the explosion was caused by the

oxygen passing through a metal tube to the ether container or by a spark from the patients' trolley. The expert also suggested that by bad luck the anæsthetist had attained the highest explosive mixture of ether and oxygen in the container—roughly 75 per cent of oxygen and 5 per cent of ether. The risks were divisible into two classes: (1) those resulting from man-made appliances, and (2) those due to the operation of the forces of Nature. Included in the first category were the modern flame and faulty electrical construction. In the latter an important factor was a static charge of electricity, a serious danger when inflammatory, volatile substances were being used. Everyone knew of the electric discharge which resulted from stroking the back of a cat. People with a dry skin were prone to discharges of static electricity, and a crackling could be heard when they combed their hair. Discharges of static electricity occurred when there was not sufficient moisture in the air to bring about earthing. Experiment appeared to confirm the idea that explosions were due to the particles of dust or droplets of liquid which were in the path of the electric current or discharge; the electric tension was ready to exhaust itself at the first opportunity. Such a source of electric discharge might well be the rubbing of the covering of the gas bag on the rubber of the bag itself; even a change in the position of the apparatus might suffice for this. To prevent such occurrences it seemed that two essentials were to increase the humidity of the air of the operating room or theatre, and to secure a proper earthing so that the static risk was reduced to a negligible quantity. It was most difficult to get a static spark in wet weather, and easiest in dry, frosty weather. The relative humidity of many operating rooms had been taken and was found to be very low, largely because of the high temperatures to which these rooms were heated, and the rapid drying of the air introduced from outside. The addition to the air of a spray or a jet of steam was recommended, with provision against the condensation of this

moisture. If the relative humidity of an operating theatre, at a temperature of 70°F., was 55, there was no guarantee against static discharges. Faraday House testing laboratories reported that, as indicated by the electroscope, the metal arms of the controls could easily be charged by merely drawing rubber blankets over the rubber mattress, but these discharges were dissipated almost at once. The metal connecting chains should be duplicated, and might with advantage be made of stainless steel or be chromium-plated.

Professor G. I. FINCH read a paper, reinforced with the assistance of Dr. Townend by a few experiments, on electrical ignition of gases. He said he proposed to deal mainly with the accidental ignition of explosive mixtures by the discharge of static electricity, or by low-tension-arc discharges, such as could arise from the rupture of a heating or an illuminating circuit. Explosion limits varied greatly according to the nature of the combustible and the supporting atmosphere, whether that were air, oxygen, or nitrous oxide. In the case of methane and air the explosion limits were found to be between 5 and 14 per cent; with a methane and oxygen mixture the variation was a very wide one—namely, 5 to 60 per cent. Little was known of the explosion limits of nitrous oxide, but this gas was an even more generous supporter of combustion than oxygen, as it was an endothermic compound, and the oxygen liberated when it broke up was very active. The extent of the explosion limit range was a measure of the ease with which mixtures exploded. Though ether-air mixtures were, under normal conditions, only explosive in the range of 1.5 to 7.5 per cent of ether in air, yet mixtures of ether in air could propagate a cool flame, which travelled slowly and could not be properly detected except in a darkened room. It was quite capable of bringing about an explosion. Hence the cool ether flame was very insidious, especially as its presence was not declared by its effect on

the skin. [This cool flame was admirably demonstrated in a twelve-foot tube of two inches diameter, and its slow progress along the tube was very evident in the darkened room.] Although the nitrous oxide mixture for anæsthesia practically placed it outside the explosion limit, it was impossible, in practice, to avoid subsequent dilution of it with air, and this dilution converted it into an explosive mixture. In order to ignite a mixture it was necessary to build up, at some point in the mixture, a sufficient concentration of suitably excited molecules, and this necessary energy was supplied by a flame, a hot body, or an electric discharge. The ignitability of an explosive mixture depended not only on the constitution of the mixture, but also on the relative proportions of the materials. [Professor Finch demonstrated this by applying the glowing end of a cigarette to them.] When an electric circuit was broken, and was stored in an inductance or a battery, it took the form of a prolonged low-tension-arc discharge. In a dry atmosphere, a man wearing rubber shoes might be charged up to a potential of several thousands of volts, and in that case there was set up a virtual inductance circuit charged with a high voltage, which might be as much as 100 amperes. [This Professor Finch illustrated by standing on an insulated mat, receiving a small charge from a Wimshurst machine, and lighting petrol with his cigarette case.] He said that static discharges could be prevented by ensuring that all bodies which could come into contact with each other should only be allowed to do so when they were of equal potential; this, in an operating theatre, called for effective earthing of people and objects and controls.

Dr. C. D. HADFIELD said that the Home Office and the Ministry of Health had approached the Medical Research Council and asked for its assistance towards undertaking a thorough investigation into the matter, so as to take whatever steps might be found necessary to prevent a recurrence of the incident. It was especially important to remember

the property of ready ignition of nitrous oxide, as there had been a tendency on the part of medical men to regard this as an inert substance from the ignition and explosion point of view.

Mr. G. E. Bell (National Physical Laboratory) alluded to parallel dangers in some industries, such as dry-cleaning. The apparatus used in the operating theatre should be kept sufficiently damp; this precaution prevented sparking in dry-cleaning processes. Equalizing of potentials could be largely brought about by earthing, and matters would be helped by getting rid of highly insulating materials: rubber tubing, rubber mats, etc., on the controls.

THE ASSOCIATION OF ANÆSTHETISTS

TWO important matters have been the chief concern of the Council of the Association since our last issue. These are the Departmental Committee which has been set up by the Government to inquire into the procedure of coroners' courts, and the negotiations towards the establishment of a Diploma in Anæsthetics. With regard to the former we are not in a position to report further than the statement that the Association has received an intimation from the Home Secretary that its willingness to appear before the Committee will not be disregarded. Regarding the Diploma, we are informed that much progress has been made in consequence of several meetings of the Association's delegates with those of the Royal Colleges and of the Committee of Management of the Examining Board. We are told that almost complete unanimity of opinion has now been reached regarding all the important points of the proposed schedule of the diploma, and we may therefore hope with confidence that before the year is out our wished-for Diploma will be established under the ægis of the two Royal Colleges. When that desideratum is reached we shall have the pleasure of putting the full details in possession of our readers. Whatever else the Association may or may not achieve in the future it will have fully justified its existence if it will be the instrument to have brought into being a satisfactory Diploma in Anæsthetics, a boon of inestimable value to the medical and general public as well as to our own specialty. Already inquiries have reached us about the Diploma from persons in widely separated parts of the Empire, and in our current correspondence readers will find a letter on the subject from a Scottish anæsthetist.

ABSTRACTS

Influence of avertin anæsthesia on epinephrine secretion. SATO, SATOW, and DEGTI in *Tohoku Journal of Experimental Medicine.* November 1934 and January 1935.

A number of experimental observations have been made on dogs. The first finding was that avertin did not interfere with the accelerating action of insulin on epinephrine secretion. Further experiments were made in which morphia played a part. These demonstrated that increase of epinephrine and of sugar due to morphia continued unabated when the animals were under the influence of avertin.

Convulsions during ether anæsthesia. E. A. ROVENSTINE in *Anæsthesia and Analgesia.* January-February 1935, p. 40.

The author seeks to correlate the symptom with the operative procedure. That this could produce the phenomenon irrespective of the anæsthetic is, he says, plausible. It is, of course, common knowledge that no theory hitherto advanced in explanation of these convulsions is in the least convincing. In support of his own, the author cites an instance. The patient was a female child of ten years, with double congenital dislocation of hip. Operation was performed under gas and oxygen, and after, morphia and scopolamine. It had been in progress over an hour when a severe convulsive seizure lasting for eight minutes took place. Operation was finished without further anæsthetic. Recovery was facilitated by intravenous glucose and six hours' oxygen insufflation. Six months later a second operation, lasting an hour, was attended by no unusual symptom. The anæsthetic was the same as before. It was repeated for a short operation a month later, and this time

tetanic contractions but no general convulsion appeared. A few days later increased traction without anæsthetic set up a convulsive seizure lasting a minute. Three more seizures of shorter duration followed in the course of the next few hours. The author gives his view that the phenomena shown by this patient suggest that convulsions may sometimes be due to trauma.

REVIEWS

Alcohol and Anæsthesia. W. BURRIDGE, D.M., M.A. Williams and Norgate, Ltd., London; pp. 62, price 2/6.

This little work contains a good deal about alcohol but little about anæsthesia. It is an interesting little philosophical treatise, the main thesis of which is that current views on the action of alcohol are necessarily wrong because the accepted physiology of stimulation and of cerebral action is also wrong. The author maintains that it is proved that there are two sources of nerve or of muscle energy, that due to altered colloidal aggregation and that due to adsorption. These two "kinesiphores" may be stimulated at the same time by one drug, and the result will be, as he maintains it is with alcohol, both exaltation and depression. Another essential feature of his doctrine is rhythmic function of neurol structures. Anæsthesia in the author's view is an alteration of balance of neurotic function, one kind of energy being increased to the detriment of the other. "General anæsthesia is a condition of feverish activity of central neurones and end organs with an altered balance of kinesiphores, instead of the one of generally depressed activity hitherto assumed." A rise of temperature, he maintains, could produce effects similar to those produced by anæsthetics, and this is obviously correct. The author's conclusions with regard to alcohol are in complete accordance with common sense whether his theories are right or wrong.

Handbook of Anæsthetics. J. STUART ROSS and H. P. FAIRLIE. Fourth edition, pp. 299, many illustrations and tables. Price 7/6. Edinburgh: E. and S. Livingstone.

Since its first appearance this has been one of the best small books on anæsthetics. The high value of the work is fully maintained in the present edition, for which Dr. Fairlie

is solely responsible. He has brought the information up to date, although the very latest additions to anæsthetics, cyclopropane and vinesthene, have not been in time to be included. The writing is clear and concise, but we deplore the use of "cyanose" as a transitive verb. The author has wisely, in our opinion, sacrificed the space demanded by numerous accounts and illustrations of various apparatus, and employed it rather in good descriptions of the general principles and practice involved in different methods of administration of anæsthetics. There is a brief but adequate account of the physiology of anæsthesia, though here we miss any reference to the carotid sinus, the importance of which in this connexion has lately been demonstrated. When discussing the choice of anæsthetic we wish the author had been more explicit with regard to abdominal operations. He states well and clearly the aims to be achieved and the general principles by which the anæsthetist is to be guided, but he does not specify the steps to be taken or the drugs or methods to be chosen. Generally speaking, the author relies in the main on ether for major surgery, and perhaps understates the value of gas and oxygen following basal anæsthetics. Since, however, the book is not intended primarily for the specialist, this cannot be reckoned a fault. For students and those practitioners who only occasionally give anæsthetics we know no better handy guide than this excellent little book.

THE SURGICAL INSTRUMENT MANUFACTURERS' ASSOCIATION (INCORPORATED)

6 Holborn Viaduct,
London, E.C. 1.

19th March, 1935.

Sir,—The Surgical Instrument Manufacturers' Association has received a communication from Sir Francis Shipway calling attention to the fact that during anæsthesia induced by a warm ether apparatus a fatality recently occurred, owing to the bellows having been connected to the wrong (or outlet) tube of the ether or chloroform bottle, with the result that liquid anæsthetic was pumped into the patient's throat. He emphasized that something must be done at once to prevent a repetition of such a disaster.

The Committee of the Surgical Instrument Manufacturers' Association are of the opinion that a question of such general importance should come within the province of the Institute of British Surgical Technicians, now in course of formation, but in view of the urgency of the matter have taken immediate action, and have designed safety valves for use with warm ether apparatus, Junker's chloroform bottles and similar anæsthetic units with which such an accident might occur in use.

The valves designed have been submitted to and approved by Sir Francis Shipway. Prints showing the exact nature and method of manufacture of these valves have been circulated to every surgical instrument firm in Great Britain affiliated to the Association. Particulars of these valves have also been circulated to the Incorporated Association of Hospital Officers and the Central Bureau of Hospital Information.

In designing these valves the Committee have borne in mind that it is necessary to render safe, in the event of incorrect fitting of bellows, the great number of existing

chloroform and ether apparatus now in use, and that this must be effected in a simple, uniform and *economical* manner.

In investigating this apparently simple problem a certain number of difficulties were encountered. Many varieties of valves were tried (glass to glass, etc.) and were found unsatisfactory. Some satisfactory valves were rejected as expensive and complicated.

Valves to use with Liquid Chloroform.

The ordinary Rigby type ball valve, fitted to the inlet tube, when immersed in chloroform was found to be sufficiently reliable to be considered safe. A ball valve fitted to the outlet tube (dry and air exposed) is not uniform in its action, is not so effective, and is not considered safe.

Valves for use with Liquid Ether.

In this case conditions are very different from those when using chloroform.

Ether is extremely mobile and penetrating. It has a low specific gravity (about half that of chloroform) and is a non-viscous fluid.

Immersing a ball valve in ether has little effect and does not improve the sealing and functioning of the valve to the extent as when it is immersed in chloroform.

Experiments show that a simple ball valve fitted into the inlet tube immersed in ether, unless it is a super-perfect valve, cannot by itself be considered safe.

A ball valve fitted to the outlet tube has the effect of making the anæsthetic bottle a closed chamber. The ether acting under the pressure of its own vapour enters the inlet tube. The method of a single ball valve on the outlet tube cannot by itself be considered safe.

It is found when using a spring-loaded ball valve on the inlet tube that if a small hole (about 1.5 mm. diameter) is drilled at the top of the inlet tube inside the bottle it is sufficient to ensure the safe working of the ball valve. On trial it is shown to prevent the ejection of ether even when the ball valve itself is not perfect.

In drawing up the recommendations noted below the Committee recognize that ball valves are not always perfect and, at times, may leak.

In the case of chloroform the fluid is heavy and is sufficiently viscous to more or less seal any slight defect.

When using ether, conditions are entirely different; there is practically no sealing.

The recommendations are as follows:

All chloroform bottles to be fitted with a ball valve of the Rigby type, spring loaded, as shown on the prints and illustrations circulated.

The inlet tube at the point where the bellows are attached should be plainly marked with a "B" or in some other unmistakable manner.

Ether Bottles, Shipway's Pattern.

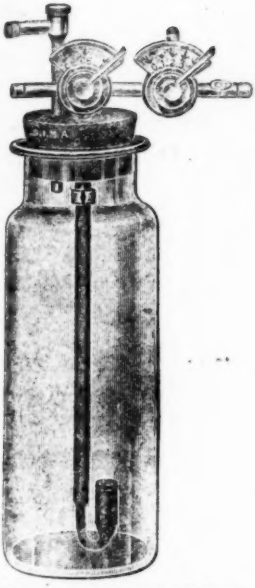
Inlet tubes to be marked as noted for chloroform bottles.

The inner tube should have a 1.5 mm. aperture at the top, inside the bottle. This aperture should be made in such a way that it is not possible for it to become accidentally covered by the rubber bung or extending tube.

The distal end of the tube which dips into the ether should be fitted with a ball valve of the Rigby type. The ball should be fixed in such a way that it cannot be accidentally displaced. It should be held down against its seating by a spring contained in the cap. This spring should be fixed to the cap or rendered inaccessible in some way so that it cannot be accidentally lost or displaced.

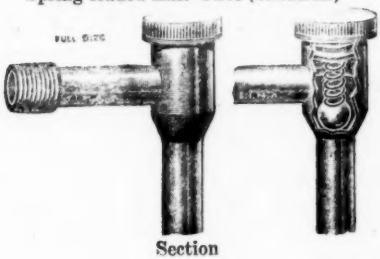
The exit tube should be furnished with a well-made valve, such as a Rigby ball valve with a spring-loading (made with the same precautions as before stated) or a flat ground copper plate valve (Major L. N. Routh's model) with a spring-loading.

When using the above valves, one point must be borne in mind. Careful examination of safety valves which have been returned for repair after prolonged use show that these valves collect sulphur and chalk from the rubber bellows, and fluff and cotton wool from the air stream. These materials become deposited in the valve; it is therefore necessary that valves should be tested periodically, and if



Shipway's (S.I.M.A.) Warm-Ether Apparatus Bottle fitted with Valves.

Spring-loaded Exit Tube (S.I.M.A.)



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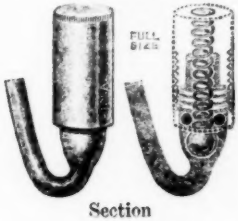
Spring-loaded (S.I.M.A.) Rigby's Valve



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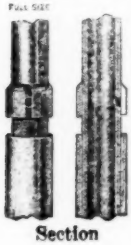
Routh's Inlet Valve (S.I.M.A.) for use with exit valve. The position of the inlet and outlet tubes should be as on the spring-loaded exit valve in other illustration

Section

Perforated Inlet Tube (S.I.M.A.)



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found not to be functioning satisfactorily they should be returned to the makers for adjustment.

General Note.

The Committee recommend all members of the Surgical Instrument Manufacturers' Association neither to stock nor list any anæsthetic apparatus with which it is possible for liquid anæsthetic to be injected into the patient's throat. Further, that if asked to make an apparatus with which such an accident is possible, they should on every occasion warn the intending purchaser of its potential dangers.

Yours faithfully,

p.p. The Surgical Instrument Manufacturers' Association,
H. GUY DREW (Down Bros., Ltd.),
Chairman.

Committee:

P. G. PHELPS (Mayer & Phelps, Ltd.) *Vice-Chairman.*
F. C. KING (Allen & Hanburys, Ltd.), *Hon. Treas.*
A. W. DOWN (Down Bros., Ltd.).
A. N. GARDNER (J. Gardner & Son),
E. G. GREVILLE (Medical Supply Association, Ltd.).
H. E. HOTCHKIN (John Weiss & Son, Ltd.).
E. W. MAYER (Mayer & Phelps, Ltd.).
T. M. PROUDFOOT (Arnold & Sons).
W. T. R. BECKETT (Beckett & Bird, Ltd.), *Hon. Sec.*

A DIPLOMA IN ANÆSTHETICS.

49 Queen's Road,
Aberdeen.
12th March, 1935.

Sir,—Your leading article in the January number of the *British Journal of Anæsthesia* on the activities of the Council of the Association of Anæsthetists of Great Britain and Ireland must have given cause for reflection to a wide circle of anæsthetists.

The consummation of the Council's task in the institution of a Diploma of Anæsthetics would involve the whole anæsthetic world in a debt of gratitude.

While it may be conceded that the requirements for a Diploma in Anæsthetics should not be such as to deter suitable candidates from the examination, it is essential that a definite and high standard must be set if the Diploma is to mark the forward step in Anæsthetics which is the aim of the Association.

The candidate for a Diploma in Anæsthetics should be a good physician who has had considerable experience of surgical work. He or she must profess the principles of regional anatomy, physiology and biochemistry, pharmacology and therapeutics, the technique of animal experiments, and the science and practice of anæsthetics in all its branches.

We anticipate that a Diploma on these lines would become a *sina qua non* to the post of honorary anæsthetist, that it would hasten the recognition of anæsthetics as a separate entity both by the medical profession and the public, and that it would lead to the provision of an anæsthetic service indispensable to the surgical unit.

Candidates should be permitted to take the examination for the Diploma two years after graduation, provided evidence is produced of considerable experience in the practice of anæsthetics or of having held the post of resident anæsthetist at a recognized hospital.

I am,

Yours sincerely,

J. Ross Mackenzie.